WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Terms	Documents
immunotoxin and CD22	57

Database: US Patents Full-Text Database
US Pre-Grant Publication Full-Text Database
JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L13

Refine Search

Recall Text | Clear

Search History

DATE: Friday, April 12, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=OR			
L13	immunotoxin and CD22	57	L13
L12	immunotoxin and (SLE or lupus).clm.	19	L12
L11	immunotoxin and (SLE or lupus)	250	L11
L10	"B cell immunotoxin" and (SLE or lupus)	0	L10
L9	L8 and ("B cell").clm.	16	L9
L8	(lupus or SLE).clm.	518	L8
L7	lupus or SLE	6158	L7
L6	L5 and (lupus or SLE)	31	L6
L5	depletion near5 "B cell"	108	L5
L4	L3 and CD22	39	L4
L3	(CD19 or CD20 or CD21 or CD23) and autoimmune	419	L3
DB=USPT,PGPB,DWPI; PLUR=YES; OP=OR			
L2	L1 and (lupus or SLE)	50	L2
L1	CD22	257	L1

END OF SEARCH HISTORY

- The Clipboard will hold a maximum of 500 items.
- Clipboard items will be lost after one hour of inactivity.

1: Behr TM, Wormann B, Gramatzki M, Riggert J, Gratz S, Behe M, Griesinger F, Sharkey RM, Kolb HJ, Hiddemann W, Goldenberg DM, Becker W. Related Articles

Low- versus high-dose radioimmunotherapy with humanized anti-CD22 or chimeric anti-CD20 antibodies in a broad spectrum of B cell-associated malignancies.
Clin Cancer Res. 1999 Oct;5(10 Suppl):3304s-3314s.
PMID: 10541379 [PubMed - indexed for MEDLINE]

2: Juweid ME, Stadtmauer E, Hajjar G, Sharkey RM, Suleiman S, Luger S, Swayne LC, Alavi A, Goldenberg DM. Related Articles

Pharmacokinetics, dosimetry, and initial therapeutic results with 131I- and (111)In-/90Y-labeled humanized LL2 anti-CD22 monoclonal antibody in patients with relapsed, refractory non-Hodgkin's lymphoma.
Clin Cancer Res. 1999 Oct;5(10 Suppl):3292s-3303s.
PMID: 10541378 [PubMed - indexed for MEDLINE]

3: Losman MJ, Hansen HJ, Dworak H, Krishnan IS, Qu Z, Shih LB, Zeng L, Goldenberg DM, Leung SO. Related Articles

Generation of a high-producing clone of a humanized anti-B-cell lymphoma monoclonal antibody (hLL2).
Cancer. 1997 Dec 15;80(12 Suppl):2660-6.
PMID: 9406722 [PubMed - indexed for MEDLINE]

4: Mattes MJ, Shih LB, Govindan SV, Sharkey RM, Ong GL, Xuan H, Goldenberg DM. Related Articles

The advantage of residualizing radiolabels for targeting B-cell lymphomas with a radiolabeled anti-CD22 monoclonal antibody.
Int J Cancer. 1997 May 2;71(3):429-35.
PMID: 9139880 [PubMed - indexed for MEDLINE]

5: Leung SO, Goldenberg DM, Dion AS, Pellegrini MC, Shevitz J, Shih LB, Hansen HJ. Related Articles

Construction and characterization of a humanized, internalizing, B-cell (CD22)-specific, leukemia/lymphoma antibody, LL2.
Mol Immunol. 1995 Dec;32(17-18):1413-27.
PMID: 8643111 [PubMed - indexed for MEDLINE]

6: Leung SO, Shevitz J, Pellegrini MC, Dion AS, Shih LB, Goldenberg DM, Hansen HJ. Related Articles, Nucleotide, Protein

Chimerization of LL2, a rapidly internalizing antibody specific for B cell lymphoma.

Hybridoma. 1994 Dec;13(6):469-76.
PMID: 7737671 [PubMed - indexed for MEDLINE]

☐ 7: Stein R, Belisle E, Hansen HJ, Goldenberg DM. Related Articles

Epitope specificity of the anti-(B cell lymphoma) monoclonal antibody, LL2.
Cancer Immunol Immunother. 1993 Oct;37(5):293-8.
PMID: 7691407 [PubMed - indexed for MEDLINE]

Display Summary Sort Save Text Clip Remove Order

Show: 20 Items 1-7 of 7 One page.

2/3/180 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

136246408 CA: 136(16)246408f PATENT
Combination therapy for treatment of autoimmune diseases using B cell depleting/immunoregulatory antibody combination.
INVENTOR(AUTHOR): Hanna, Nabil
LOCATION: USA
ASSIGNEE: Idec Pharmaceuticals
PATENT: PCT International ; WO 200222212 A2 DATE: 20020321
APPLICATION: WO 2001US29026 (20010918) *US PV257147 (20001222)
PAGES: 58 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61P-037/00; A61K-039/39 DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CO; CU; CZ; EE; ES; FI; GB; GE; GM; HU; ID; IL; IN; KP; KR; LK; LR; MN; MW; MX; NO; NZ; PL; PT; RO; RU DESIGNATED REGIONAL: GH; GM ; KE; LS; MW; MZ; SD; SL; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; MC; NL; PT; SE; TR; CG; CI; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/181 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. resery.

136068707 CA: 136(5)68707t PATENT
Treatment of B-cell associated diseases such as malignancies and autoimmune diseases using a cold anti-CD20 antibody/radiolabeled anti-CD22 antibody combination
INVENTOR(AUTHOR): White, Christine
LOCATION: USA
ASSIGNEE: Idec Pharmaceuticals Corporation
PATENT: PCT International ; WO 200197858 A2 DATE: 20011227
APPLICATION: WO 2001US18939 (20010614) *US PV212668 (20000620)
PAGES: 59 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-051/00A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/182 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

136036352 CA: 136(3)36352g PATENT
Identification of unique binding interactions between certain antibodies and the human B7.1 and B7.2 costimulatory antigens
INVENTOR(AUTHOR): Anderson, Darrell R.; Hanna, Nabil; Brams, Peter
LOCATION: USA
ASSIGNEE: Idec Pharmaceuticals Corporation
PATENT: PCT International ; WO 200189567 A1 DATE: 20011129
APPLICATION: WO 2001US16364 (20010522) *US 576424 (20000522)
PAGES: 89 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A; C07K-016/28B DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM;

AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ ; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/183 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

135302781 CA: 135(21)302781r JOURNAL
Prevention of experimental autoimmune encephalomyelitis in the common marmoset (Callithrix jacchus) using a chimeric antagonist monoclonal antibody against human CD40 is associated with altered B cell responses
AUTHOR(S): Boon, Louis; Brok, Herbert P. M.; Bauer, Jan; Ortiz-Buijsse, Antonio; Schellekens, Marc M.; Ramdien-Murli, Seema; Blezer, Erwin; Van Meurs, Marjan; Ceuppens, Jan; De Boer, Mark; 't Hart, Bert A.; Laman, Jon D.
LOCATION: Tanox Pharma B. V., Amsterdam, Neth.
JOURNAL: J. Immunol. DATE: 2001 VOLUME: 167 NUMBER: 5 PAGES: 2942-2949 CODEN: JOIMA3 ISSN: 0022-1767 LANGUAGE: English PUBLISHER: American Association of Immunologists

2/3/184 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

134041104 CA: 134(4)41104r PATENT
Immunotherapy of autoimmune disorders using antibodies which target B-cells
INVENTOR(AUTHOR): Goldenberg, David M.; Hansen, Hans J.
LOCATION: USA
ASSIGNEE: Immunomedics, Inc.
PATENT: PCT International ; WO 200074718 A1 DATE: 20001214
APPLICATION: WO 2000US15780 (20000609) *US PV138284 (19990609)
PAGES: 39 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A; C07K-016/28B; C07K-019/00B; A61P-037/06B; C07K-014/55B
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZW ; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/185 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

133361915 CA: 133(26)361915u PATENT
Treatment of autoimmune diseases with antagonists which bind to B cell surface markers
INVENTOR(AUTHOR): Curd, John G.; Kunkel, Lori A.; Grillo-Lopez, Antonio J.
LOCATION: USA
ASSIGNEE: Genentech, Inc.; Idec Pharmaceuticals, Inc.
PATENT: PCT International ; WO 200067796 A1 DATE: 20001116
APPLICATION: WO 2000US40018 (20000504) *US PV133018 (19990507) *US PV139621 (19990617)
PAGES: 34 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A; A61P-037/00B; A61K-047/48B; C07K-016/28B DESIGNATED COUNTRIES: AE; AG; AL;

AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS ; MW; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/186 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

128256389 CA: 128(21)256389z PATENT
Immune direction therapy
INVENTOR(AUTHOR): Prendergast, Patrick T.
LOCATION: Ire.,
ASSIGNEE: Prendergast, Patrick T.
PATENT: PCT International ; WO 9810787 A2 DATE: 19980319
APPLICATION: WO 97IB1086 (19970910) *US 25180 (19960911)
PAGES: 83 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/00A; C07K-016/00B DESIGNATED COUNTRIES: AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CU; CZ; DE; DK; EE; ES; FI; GB; GE; GH; HU; ID; IL; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA; UG; US; UZ; VN; YU; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; KE; LS ; MW; SD; SZ; UG; ZW; AT; BE; CH; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE; SN; TD; TG

2/3/187 (Item 8 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

128010314 CA: 128(2)10314f PATENT
Anti-CD19 and anti-CD22 monoclonal antibodies and immunotoxins and therapeutic uses thereof
INVENTOR(AUTHOR): Uhr, Jonathan W.; Vitetta, Ellen S.; Scheuermann, Richard H.
LOCATION: USA
ASSIGNEE: Board of Regents, the University of Texas
PATENT: United States ; US 5686072 A DATE: 19971111
APPLICATION: US 202042 (19940222) *US 899781 (19920617)
PAGES: 34 pp. Cont.-in-part of U.S. Ser. No. 899,781, abandoned. CODEN: USXXAM LANGUAGE: English CLASS: 424183100; A61K-039/395A

2/3/188 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0277938 DBA Accession No.: 2002-01440
Human IgG monoclonal anti-alphaIIb beta3-binding fragments derived from immunized donors using phage display - the use of monoclonal antibody
AUTHOR: Jacobin M J; Laroche-Traineau J; Little M; Keller A; Peter K; Welschof M; Nurden A; +Clofent-Sanchez G
CORPORATE AFFILIATE: CNRS
CORPORATE SOURCE: Center de la Recherche Scientific, Unite Mixte de Recherche 5533, Hopital Cardiologique, Avenue de Magellan, 33604, Pessac, France. email:gisele.clofent@unmr5533.u-bordeaux2.fr
JOURNAL: J.Immunol. (168, 4, 2035-45) 2002
ISSN: 0022-1767 CODEN: JOIMA3
LANGUAGE: English

2/3/189 (Item 2 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0247249 DBA Accession No.: 2000-01739 PATENT
Novel methods for treating lupus and reversing lupus-associated kidney disease using anti-gp39 antibodies - chimeirc antibody or humanized antibody used to prevent T-lymphocyte-mediated B-lymphocyte activation in systemic lupus erythematosus therapy
AUTHOR: Noelle R J; Burns C M
CORPORATE SOURCE: Hanover, NH, USA
PATENT ASSIGNEE: Dartmouth-Coll. 1999
PATENT NUMBER: WO 9951258 PATENT DATE: 19991014 WPI ACCESSION NO.: 1999-633696 (1954)
PRIORITY APPLIC. NO.: US 54488 APPLIC. DATE: 19980403
NATIONAL APPLIC. NO.: WO 99US7321 APPLIC. DATE: 19990402
LANGUAGE: English

2/3/190 (Item 3 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0226040 DBA Accession No.: 98-07637 PATENT
New monoclonal antibodies specific for B7.1 or B7.2 antigens and inhibiting binding to CD28 - monoclonal antibody, produced by monkey hybridoma cell culture, humanized antibody, primatized antibody and chimeric antibody, used for **autoimmune** disease therapy
AUTHOR: Anderson D R; Hanna N; Brams P
CORPORATE SOURCE: San Diego, CA, USA.
PATENT ASSIGNEE: Idec-Pharm. 1998
PATENT NUMBER: WO 9819706 PATENT DATE: 980514 WPI ACCESSION NO.: 98-286601 (9825)
PRIORITY APPLIC. NO.: US 746361 APPLIC. DATE: 961108
NATIONAL APPLIC. NO.: WO 97US19906 APPLIC. DATE: 971029
LANGUAGE: English

2/3/191 (Item 4 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0222268 DBA Accession No.: 98-03865 PATENT
Determination of lymphocyte distribution and trafficking in mammals - using monoclonal antibody- or recombinant antibody-labeled lymphocytes
AUTHOR: Rubin R H; Fischman A J; Baltimore D
CORPORATE SOURCE: Cambridge, MA, USA; Boston, MA, USA.
PATENT ASSIGNEE: Massachusetts-Inst.Technol.; Gen.Hosp.Boston 1998
PATENT NUMBER: WO 9800560 PATENT DATE: 980108 WPI ACCESSION NO.: 98-086984 (9808)
PRIORITY APPLIC. NO.: US 21083 APPLIC. DATE: 960702
NATIONAL APPLIC. NO.: WO 97US11582 APPLIC. DATE: 970701
LANGUAGE: English

2/3/192 (Item 5 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0219868 DBA Accession No.: 98-01465 PATENT
Enhancing B-cell cytotoxicity of CD22-binding immunotoxin - monoclonal antibody, Fab' and Fab'Fc fragment for use in immunotoxin for cancer or

autoimmune disease therapy
AUTHOR: Uhr J W; Vitetta E S; Scheuermann R H
CORPORATE SOURCE: Austin, TX, USA.
PATENT ASSIGNEE: Univ.Texas 1997
PATENT NUMBER: US 5686072 PATENT DATE: 971111 WPI ACCESSION NO.: 97-558086 (9751)
PRIORITY APPLIC. NO.: US 202042 APPLIC. DATE: 940222
NATIONAL APPLIC. NO.: US 202042 APPLIC. DATE: 940222
LANGUAGE: English

2/3/193 (Item 6 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0205745 DBA Accession No.: 97-00866 PATENT
Improving transfection of T cells by preliminary costimulation of proliferating cells - primary T-lymphocyte transfection method for HIV virus infection and **autoimmune** disease gene therapy
AUTHOR: June C H; Thompson C B; Kim S
CORPORATE SOURCE: Bethesda, MD, USA; Ann Arbor, MI, USA.
PATENT ASSIGNEE: U.S.Navy; Univ.Michigan 1996
PATENT NUMBER: WO 9634970 PATENT DATE: 961107 WPI ACCESSION NO.: 96-506172 (9650)
PRIORITY APPLIC. NO.: US 475136 APPLIC. DATE: 950607
NATIONAL APPLIC. NO.: WO 96US6200 APPLIC. DATE: 960502
LANGUAGE: English

2/3/194 (Item 7 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0178937 DBA Accession No.: 95-06347 PATENT
Treating a T-cell and/or B-cell mediated **autoimmune** disease - myelin oligodendrocyte glycoprotein anti-idiotype monoclonal antibody and recombinant peptide preparation for use in multiple sclerosis therapy and diagnosis
AUTHOR: Bernard C C A; Kerlero de Rosbo N C M
PATENT ASSIGNEE: Univ.La-Trobe 1995
PATENT NUMBER: WO 9507096 PATENT DATE: 950316 WPI ACCESSION NO.: 95-123238 (9516)
PRIORITY APPLIC. NO.: AU 931030 APPLIC. DATE: 930906
NATIONAL APPLIC. NO.: WO 94AU522 APPLIC. DATE: 940902
LANGUAGE: English

2/3/195 (Item 8 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0177776 DBA Accession No.: 95-04597 PATENT
New monoclonal antibody recognizing CDIM epitope on B-cells - produced by hybridoma cell culture, for use in cancer or **autoimmune** disease therapy, or as a diagnostic agent
AUTHOR: Bhat N M; Bieber M M; Teng N H
PATENT ASSIGNEE: Univ.Leland-Stanford-Jr. 1995
PATENT NUMBER: WO 9503770 PATENT DATE: 950209 WPI ACCESSION NO.: 95-081998 (9511)
PRIORITY APPLIC. NO.: US 101436 APPLIC. DATE: 930802
NATIONAL APPLIC. NO.: WO 94US8793 APPLIC. DATE: 940802
LANGUAGE: English

2/3/196 (Item 9 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0163340 DBA Accession No.: 94-05891 PATENT
Novel B-lymphoma cell line and purified antigen - new interleukin-6-dependent B-lymphocyte lymphoma DS1 cell line; monoclonal antibody production; potential vaccine production and **autoimmune** disease therapy and diagnosis
PATENT ASSIGNEE: U.S.Dept.Health-Human-Serv. 1994
PATENT NUMBER: WO 9404659 PATENT DATE: 940303 WPI ACCESSION NO.: 94-083179 (9410)
PRIORITY APPLIC. NO.: US 934106 APPLIC. DATE: 920821
NATIONAL APPLIC. NO.: WO 93US7856 APPLIC. DATE: 930820
LANGUAGE: English

2/3/197 (Item 10 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0128564 DBA Accession No.: 92-01056 PATENT
Monoclonal antibody and immunoconjugates - MAb J3-119 produced by hybridoma ATCC HB-10383; application to B-lymphocyte disorder therapy, e.g. leukemia and **autoimmune** disease e.g. rheumatoid arthritis
PATENT ASSIGNEE: Biomembrane-Inst. 1991
PATENT NUMBER: WO 9113974 PATENT DATE: 910919 WPI ACCESSION NO.: 91-295629 (9140)
PRIORITY APPLIC. NO.: US 560154 APPLIC. DATE: 900731
NATIONAL APPLIC. NO.: WO 91US1649 APPLIC. DATE: 910312
LANGUAGE: English

2/3/198 (Item 11 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0120038 DBA Accession No.: 91-07680 PATENT
Treating **autoimmune** disease associated with particular immunoglobulin isotypes - B-lymphocyte-bound IgM, IgG migis epitope mouse monoclonal antibody, chimeric antibody, bispecific antibody production; hybridoma, transfectoma construction; DNA sequence
PATENT ASSIGNEE: Tanox-Biosyst. 1991
PATENT NUMBER: WO 9104055 PATENT DATE: 910404 WPI ACCESSION NO.: 91-117334 (9116)
PRIORITY APPLIC. NO.: US 408123 APPLIC. DATE: 890915
NATIONAL APPLIC. NO.: WO 90US5229 APPLIC. DATE: 900914
LANGUAGE: English

2/3/199 (Item 12 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0072254 DBA Accession No.: 88-03103 PATENT
New B-lymphocyte surface antigen Bp50 and ligands binding to it - especially monoclonal antibody G28-5 produced by a hybridoma; immunostimulant activity
PATENT ASSIGNEE: Oncogen 1987
PATENT NUMBER: GB 2191494 PATENT DATE: 871216 WPI ACCESSION NO.: 87-350452 (8750)
PRIORITY APPLIC. NO.: US 3884 APPLIC. DATE: 860613
NATIONAL APPLIC. NO.: GB 8713650 APPLIC. DATE: 870611
LANGUAGE: English

2/3/200 (Item 13 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0047432 DBA Accession No.: 86-05280
Human monoclonal antibodies as probes to study **autoimmune** and allergic disorders - methods of human hybridoma and monoclonal antibody preparation and applications in diagnosis, therapy etc.
AUTHOR: Chiorazzi N
CORPORATE SOURCE: The Rockefeller University, 1230 York Avenue, New York, New York 10021, U.S.A.
JOURNAL: Bio/Technology (4, 3, 210-18) 1986
CODEN: 2049Y
LANGUAGE: English
?

2/3/190 (Item 3 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0226040 DBA Accession No.: 98-07637 PATENT
New monoclonal antibodies specific for B7.1 or B7.2 antigens and inhibiting binding to CD28 - monoclonal antibody, produced by monkey hybridoma cell culture, humanized antibody, primatized antibody and chimeric antibody, used for **autoimmune** disease therapy
AUTHOR: Anderson D R; Hanna N; Brams P
CORPORATE SOURCE: San Diego, CA, USA.
PATENT ASSIGNEE: Idec-Pharm. 1998
PATENT NUMBER: WO 9819706 PATENT DATE: 980514 WPI ACCESSION NO.: 98-286601 (9825)
PRIORITY APPLIC. NO.: US 746361 APPLIC. DATE: 961108
NATIONAL APPLIC. NO.: WO 97US19906 APPLIC. DATE: 971029
LANGUAGE: English

plasma cells. Analogous to their normal **B-cell** counterparts, **CD20** is expressed on malignant lymphoplasmacytic cells from most patients with Waldenstrom's macroglobulinemia and on malignant plasma cells from a fraction (20%) of **multiple** myeloma patients. **CD20** also is expressed on subpopulations of normal donor plasma cells, which may include autoantibody-secreting plasmacytes. In view of these findings, the anti-**CD20** chimeric monoclonal **antibody**, rituximab (Rituxan; Genentech, Inc, South San Francisco, CA and IDEC Pharmaceutical Corporation, San Diego, CA), has been evaluated in the treatment of Waldenstrom's macroglobulinemia and **multiple** myeloma, as well as in nonmalignant plasma cell disorders including IgM polyneuropathies, immune thrombocytopenias, and **autoimmune** hemolytic anemias, with reported activity in these entities. An update of these clinical efforts is presented in this report.

2/7/105 (Item 33 from file: 73)
DIALOG(R)File 73:EMBASE

06499324 EMBASE No: 1996166364
The fate of self-reactive B cells depends primarily on the degree of antigen receptor engagement and availability of T cell help
Fulcher D.A.; Lyons A.B.; Korn S.L.; Cook M.C.; Koleda C.; Parish C.; De St. Groth B.F.; Basten A.
Immunology Unit, Inst. of Clinical Pathol./Med. Res., Westmead Hospital,Sydney, NSW 2145 Australia
Journal of Experimental Medicine (J. EXP. MED.) (United States) 1996, 183/5 (2313-2328)
CODEN: JEMEA ISSN: 0022-1007
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

Self-reactive B cells from tolerant double-transgenic (Dbl-Tg) mice coexpressing hen egg lysozyme (HEL) and rearranged anti-HEL immunoglobulin genes have a relatively short life span when compared to normal B cells, irrespective of whether they are exposed to antigen in multivalent membrane- bound form (mHEL-Dbl-Tg mice) or soluble form (sHEL-Dbl-Tg mice). The factors responsible for determining the fate of these B cells after encounter with self-antigen were investigated using a cell tracking technique in which anti- HEL Ig-Tg spleen cells were labeled with the intracellular dye 5- carboxyfluorescein diacetate-succinimidyl ester (CFSE) and injected either into non-Tg recipients or a variety of HEL-Tg hosts. In non-Tg recipients, HEL-binding B cells persisted in the circulation and could be detected in the follicles of the spleen for at least 5 d. On transfer into either mHEL-Tg or sHEL-Tg hosts, they underwent activation and then rapidly disappeared from the blood and spleen over the next 3 d, consistent with the short life span reported previously. Immunohistology of spleens from sHEL-Tg recipients indicated that the transferred B cells had migrated to the outer margins of the periarteriolar lymphoid sheath (PALS), where they were detectable for 24 h before being lost. The positioning or B cells in the outer PALS depended on a critical threshold of Ig receptor binding corresponding to a serum HEL concentration between 0.5 and 15 ng/ml, but was not restricted to endogenously expressed HEL in that the same migratory pattern was observed after transfer into non-Tg recipients given exogenous (foreign) HEL. Moreover, bone marrow-derived immature Ig-Tg B cells horned to the outer PALS of sHEL-Tg mice and then disappeared at the same rate as mature B cells, indicating that the stage of maturation did not influence the fate of self- reactive B cells m a tolerant environment. On the other hand, HEL-binding B cells transferred into sHEL-Dbl-Tg recipients persisted over the 3-d period of study, apparently due to insufficient availability of antigen, as indicated by the fact that the degree of Ig receptor downregulation on the transferred B cells was much less than in sHEL-Tg recipients. If T cell help was provided to Ig Tg

B cells at the time of transfer into sHEL-Tg recipients in the form of preactivated CD4sup + T cells specific for major histocompatibility complex-peptide complexes on the **B cell** surface, HEL- binding **B cells** migrated through the outer PALS of the spleen to the follicle, where they formed germinal centers, or to adjacent red pulp, where they formed proliferative loci and secreted significant amounts of anti-HEL **antibody**. Taken **together**, these results indicated that the outcome of the interaction between self-antigen and **B cells** is largely determined by a **combination** of the degree of receptor engagement and availability of T cell help.

2/7/109 (Item 37 from file: 73)
DIALOG(R)File 73:EMBASE

06314907 EMBASE No: 1995352867
The human B-cell repertoire: Expression in normal and pathological conditions
LE REPERTOIRE B HUMAIN: EXPRESSION NORMALE ET PATHOLOGIQUE
Huck S.; Zouali M.
Lab. d'immunogenetique moleculaire, UMR9942, IGMM, 1919 route de Mende,34033 Montpellier Cedex 01 France
Medecine/Sciences (MED. SCI.) (France) 1995, 11/11 (1566-1575)
CODEN: MSMSE ISSN: 0767-0974
DOCUMENT TYPE: Journal; Review
LANGUAGE: FRENCH SUMMARY LANGUAGE: FRENCH; ENGLISH

The timing and mechanisms that govern B-cell development are unique in several respects. Immunoglobulin (Ig) genes are exquisitely assembled in B cells and this process is site-specific. In order to express Ig on their surface, B-cell progenitors must rearrange variable (V) gene segments on the Ig heavy (H), and kappa and lambda light (L) chain loci in a process known as VDJ recombination. During human ontogeny, B-cell progenitors can choose from the libraries of variable gene segments. This primary repertoire is further expanded by junctional flexibility, secondary rearrangements, i.e. of VL to VL and somatic hypermutation. In human, the organization and the content of the Ig loci have been the focus of considerable attention. The recent elucidation of the structure of the VH and Vkappa loci revealed that human Ig V genes exhibit unique features. For both loci, the V genes have been categorized into families of sequence-related members which are intermingled among each other and include approximately 45% of pseudo-genes. Even more intriguing are the observations that some VH and Vkappa genes are dispersed not only outside the functional loci, but also on other chromosomes. The molecular events that gave rise to these 'orphon' genes underlie the extensive degree of plasticity of human Ig genes. While in normal conditions, they could play a role in amplifying the repertoire, in pathological conditions they may in turn become the targets for chromosomal translocations in B-cell neoplasia. Other remarkable features of the human VH locus include its dominance by members of the VH3 gene-family and its high degree of polymorphism, with insertion deletion/polymorphisms and allelisms, which may lead to a high number of haplotypes with different coding potentials. The possibility that certain polymorphisms are associated with disease susceptibility has not been fully addressed. The elucidation of the content of the human Ig V loci is providing insight into the mechanisms of V gene expression in pathological conditions. A number of studies focused on V gene usage in relation to systemic **autoimmunity**. In principle, random **combinatorial** assembly of Ig V gene elements may lead to generation of a proportion of **antibodies** with high-affinity to self. In normal conditions, the corresponding **B cells** will be clonally deleted, rendered silent, or switched to a different specificity, a mechanism that has been termed receptor editing. In systemic **autoimmunity**, however, anti-self high-affinity antibodies are overexpressed. Molecular cloning,

nucleotide sequencing and comparative genomic PCR revealed that human pathogenic autoantibody V genes have incurred extensive somatic selection events. There is also evidence that the corresponding B cells underwent essentially primary L-chain rearrangements. This observation raises the possibility that, in systemic **autoimmunity**, a subset of B cells may be unable to revise their receptors and to extinguish their high-affinity for self. This blockade could be genetically determined or somatically acquired. Finally, infection with the human immunodeficiency virus (HIV) results in several B cell abnormalities. The first evidence that HIV antigens may alter repertoire expression came from the demonstration that the major envelope protein of HIV-1, gp120, binds to V(H)3sup + B cells and to serum V(H)3sup + Ig from normal individuals, and this interaction is independent of the L-chain isotype. The gp120 interaction with V(H)3 gene products also seems to be functional because gp120 selectively induces Ig secretion by V(H)3 B cells. These characteristics are reminiscent of the properties of B-cell superantigens which are able to trigger a large proportion of the B-cell repertoire. More recently, studies of peripheral blood lymphocytes from HIV seropositive subjects and AIDS patients show marked changes in V(H)3sup + B cells during different clinical stages of HIV infection. It is possible that the sequential expansion and reduction of V(H)3 B cells are related to the superantigenic properties of gp120. Understanding the molecular basis of this activity and the cellular mechanisms responsible for the clonal fate of B cells during HIV infection are relevant to designing novel strategies for immunointervention.
?

ds

Set Items Description
S1 302 (B(W)LYMPHOCYT? OR B(W)CELL? OR CD20 OR CD22) (20N)(COMBIN? OR TOGETHER OR MULTIPLE)(20N)(ANTIBOD?) AND AUTOIMMUN?
S2 200 RD S1 (unique items)
? t s2/7/24,82,105,109

2/7/24 (Item 24 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12563474 BIOSIS NO.: 200000316976
The role of B cells and autoantibodies in multiple sclerosis.
AUTHOR: Archelos Juan J; Storch Maria K; Hartung Hans-Peter
AUTHOR ADDRESS: (a)Multiple Sclerosis Research Group, Department of Neurology, Karl-Franzens-Universitat, Auenbruggerplatz 22, A-8036, Graz** Austria
JOURNAL: Annals of Neurology 47 (6):p694-706 June, 2000
MEDIUM: print
ISSN: 0364-5134
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: A variety of cellular and humoral immunological abnormalities have been observed in **multiple** sclerosis (MS). In the past few years, several lines of evidence converged to imply an important role of autoreactive **antibodies** and **B cells** in the pathogenesis of MS. Recent data suggest that autoantibodies may be harmful in lesion formation but also potentially beneficial in repair. This review surveys recent advances in the concepts of generation and nature of pathogenetic autoantibodies, their potential modes of action, mechanisms of their long-term persistence, and the role of the inflamed brain tissue as a B-cell-supporting microenvironment in MS. Based on the presence of specific autoantibodies, it seems possible to define distinct MS subgroups in the near future. The therapeutic relevance of these new findings is presented.

2/7/82 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE

11040675 EMBASE No: 2001073339
The use of rituximab in the treatment of malignant and nonmalignant plasma cell disorders
Treon S.P.; Anderson K.C.
Dr. S.P. Treon, Department of Adult Oncology, Dana Farber Cancer Institute, 44 Binney St, Boston, MA 02115 United States
Seminars in Oncology (SEMIN. ONCOL.) (United States) 2000, 27/6 SUPPL. 12 (79-85)
CODEN: SOLGA ISSN: 0093-7754
DOCUMENT TYPE: Journal ; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 49

CD20 is a B-cell-restricted antigen that, for the most part, is expressed from the pre-B-cell to the mature B-cell stage of B-cell differentiation. Several transcription factors regulate CD20 expression during B-cell differentiation, the most important of which appear to be PU.1 and Pip (PU.1 interacting protein). As B cells differentiate to plasma cells, CD20 expression is downregulated, which coincides with PU.1 downregulation in

2/3/123 (Item 51 from file: 73)
DIALOG(R)File 73:EMBASE

03952214 EMBASE No: 1989121207
B cells and antibodies in MS
Link H.; Baig S.; Jiang Y.-P.; Olsson O.; Hojeberg B.; Kostulas V.; Olsson T.
Department of Neurology, Karolinska Institutet, Huddinge University Hospital, S-141 86 Stockholm Sweden
Research in Immunology (RES. IMMUNOL.) (France) 1989, 140/2 (219-226)
CODEN: RIMME ISSN: 0923-2494
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

```
Set     Items   Description
S1        637   (CD20 OR B1) (20N)(CD22 OR LYB8 OR BL(W)CAM)
S2          0   (CD20 OR B1) (20N)(CD22 OR LYB8 OR BL(W)CAM)(20N)(ANTIBOD$-
              )(20N)(COMBIN? OR TOGETHER OR BOTH)
S3          0   (CD20 OR B1) (20N)(CD22 OR LYB8 OR BL(W)CAM)(20N)(ANTIBOD$)
              AND (ANTIBOD$)(20N)(COMBIN? OR TOGETHER OR BOTH OR MIX?)
S4          0   (CD20 OR B1) (20N)(CD22 OR LYB8 OR BL(W)CAM)(20N)(ANTIBOD$)
S5          0   (CD20 OR B1) (20N)(CD22 OR LYB8 OR BL(W)CAM) AND (ANTIBOD$)
S6        637   (CD20 OR B1) (20N)(CD22 OR LYB8 OR BL(W)CAM)
S7        173   (CD20 OR B1) (20N)(CD22 OR LYB8 OR BL(W)CAM)(20N)(ANTIBOD?)
S8         64   S7 AND (ANTIBOD?)(20N)(MIX? OR COMBIN? OR TOGETHER OR BOTH
              OR TWO)
S9         41   RD S8 (unique items)
?
```

13632819 BIOSIS NO.: 200200261640

Combination monoclonal **antibody** therapy for lymphoma: Treatment with epratuzumab (anti-**CD22**) and rituximab (anti-**CD20**) is well tolerated.

AUTHOR: Leonard John P(a); Coleman Morton(a); Matthews Jamie C(a); Fiore Jennifer M(a); Dosik Alan(a); Kapushoc Heather; Kin Elizabeth; Cesano Alessandra; Wegener William A; Goldenberg David M

AUTHOR ADDRESS: (a)Center for Lymphoma and Myeloma, Division of Hematology/Oncology, Weill Medical College of Cornell University and New York Presbyterian Hospital, New York, NY**USA

JOURNAL: Blood 98 (11 Part 1):p844A November 16, 2001

MEDIUM: print

CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001

ISSN: 0006-4971

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Epratuzumab, a humanized monoclonal antibody directed against the B-cell specific antigen CD22, has been demonstrated to be a safe and active monotherapy in phase I/II trials for the treatment of relapsed and refractory non-Hodgkin's lymphoma (NHL). Given the potential for synergy with other biologic agents, we have conducted a phase II trial to examine the safety and efficacy of the **combination** of epratuzumab with rituximab, a chimeric anti-CD20 **antibody**, for the treatment of NHL. Eighteen patients have been enrolled and treated on study, 12 of which are currently evaluable for safety only. Half of evaluable patients were over age 60 (range 21-84) and 50% had received 2 or more prior regimens (range 1 to 5). All patients were rituximab-naive. NHL histologies include diffuse large B cell (3), follicular (8), and marginal zone (1). The treatment regimen included 4 weekly infusions of epratuzumab (360 mg/m2/week over 60 min) and rituximab (375 mg/m2/week over 4-6 hrs). Acetaminophen and diphenhydramine premedication was administered, and in the initial cohort (6 pts), epratuzumab was administered on day 1, rituximab on day 3, and both drugs on days 8, 15, 22. In cohort 2, both drugs (epratuzumab followed by rituximab) were provided on days 1, 8, 15, 22. Most treatment-related adverse events occurred with the first week's infusion, and all were grade 1-2. No infusion reaction required termination of infusion, and no drug-related SAEs were observed. The most frequently reported adverse events were chills/rigors (5/12, 42%), fever (4/12, 34%), nausea and flushing (3 subjects each, 25%). Other less common treatment-related adverse events (reported in 1 or 2 subjects only) were abdominal pain, dyspnea, fatigue, headache. One subject who received the week 1 infusions separated by 48 hours had no adverse events. The majority of infusion-related events appeared to be temporally related to rituximab infusion, although with the sequential nature of the treatment, drug attribution is unclear. Given the delayed time to response with biologic agents, efficacy results are premature with a limited number of evaluable patients, but to date 5 objective responses have been observed, all of which are CRs. These preliminary data demonstrate that the addition of epratuzumab into a **combination** regimen does not appear to alter the known safety profile of rituximab. This **combination** monoclonal **antibody** therapeutic regimen appears feasible, has promising activity, and warrants further evaluation in larger clinical studies in NHL.

9/7/2 (Item 2 from file: 5)

13612370 BIOSIS NO.: 200200241191

9/7/38 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07011540 91323796 PMID: 1864552

Immunomagnetic purging procedure for autologous bone marrow transplantation in lymphoid malignancies.

De Rosa L; Montuoro A; Pandolfi A; Lanti T; Pescador L; Morara R; De Laurenzi A

Divisione di Ematologia, Ospedale San Camillo, Roma, Italy.

Haematologica (ITALY) Mar 1991, 76 Suppl 1 p37-40, ISSN 0390-6078 Journal Code: 0417435

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

In this report we describe the use of the immunomagnetic depletion of tumor cells from harvested bone marrow in two patients with acute lymphoblastic leukemia. The immunomagnetic procedure of purging involved one incubation of the marrow cells with a **mixture** of 4 monoclonal **antibodies** which bind to antigens of CD10 (ALB2), CD19 (HD37), **CD20** (**B1**) and **CD22** (HD39). **Two** incubations with magnetic beads (Dynabeads M-450) covered with antimouse **antibodies** followed by magnetic separation were performed. The bead/target B cell ratio was 50:1. After purging the recovery of mononuclear cells was 56% and 40%, while the recovery of CFU-GM was 45% and 38% respectively. Both patients engrafted rapidly without serious complications. One patient relapsed 4 months after transplant, the other remains in complete remission after 5 months. Our results confirm that the use of immunomagnetic beads is a simple, safe and reproducible technique to remove tumor cells before ABMT in patients with B malignancies using a broad mixture of MoAbs. However only a randomized trial using autologous marrow purged or not will clarify the effective clinical value of the procedure.

Record Date Created: 19910909

05636251 BIOSIS NO.: 000083109396
VALUE OF MONOCLONAL ANTI-CD22 P135 ANTIBODIES FOR THE DETECTION OF NORMAL AND NEOPLASTIC B LYMPHOID CELLS
AUTHOR: MASON D Y; STEIN H; GERDES J; PULFORD K A F; RALFKIAER E; FALINI B; ERBER W N; MICKLEM K; GATTER K C
AUTHOR ADDRESS: HAEMATOLOGY DEPARTMENT, JOHN RADCLIFFE HOSPITAL, OXFORD, OX39DU, ENGLAND.
JOURNAL: BLOOD 69 (3). 1987. 836-840. 1987
FULL JOURNAL NAME: Blood
CODEN: BLOOA
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: **Two** monoclonal **antibodies** (To15 and 4KB128) specific for the B cell-associated CD22 antigen (135,000 mol wt) are described. On immunoenzymatic analysis of cryostat tissues sections, these **antibodies** strongly label **both** mantle zone and geminal center B lymphoid cells in secondary lymphoid follicles (and also scattered extrafollicular lymphoid cells) but are unreactive with other cell types (with the exception of weak reactivity with some epithelioid histiocytes). These reactions differ from those of monoclonal **antibodies** B1 and B2 (anti-**CD20** and CD21) but are similar to those of the pan-B **antibody** B4 (anti -CD19). One of the anti-CD-22 **antibodies** (To15) has been tested extensively by immunoenzymatic labeling on < 350 neoplastic lymphoid and hematological samples. The **CD22** antigen was found in tissues sections in most B cell-derived neoplasms, the major exceptions being myeloma (all cases negative) and a small proportion of high-grade lymphoma (6% of cases negative). In cell smears, the antigen could be found on neoplastic cells in most B cell lymphoproliferative disorders, including common acute lymphoblastic leukemia (ALL) (90% positive) and B cell chronic lymphocytic leukemia (CLL) (89% positive). We conclude that anti-CD22 antibodies are of value for identification of human B cell lymphoproliferative disorders (especially when used in conjunction with anti-CD19 antibodies). Previous reports that the CD22 antigen is absent from many B cell neoplasms are probably due to its being expressed within the cytoplasm of immature B cells rather than on their surface.

06619614 BIOSIS NO.: 000087061776
THE EPITOPE SPECIFICITY AND TISSUE REACTIVITY OF FOUR MURINE MONOCLONAL ANTI-CD22 ANTIBODIES
AUTHOR: LI J-L; SHEN G-L; GHETIE M-A; MAY R D; TILL M; GHETIE V; UHR J W; JANOSSY G; THORPE P E
AUTHOR ADDRESS: DEP. MICROBIOL., UNIV. TEX. SOUTHWESTERN MED. CENT. DALLAS, DALLAS, TEX. 75235.
JOURNAL: CELL IMMUNOL 118 (1). 1989. 85-99. 1989
FULL JOURNAL NAME: Cellular Immunology
CODEN: CLIMB
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: The CD22 antigen is expressed on the surface of normal human B cells and some neoplastic B cell lines and tumors. Previous cross-blocking studies using a panel of monoclonal anti-CD22 antibodies have defined four epitope groups, termed A-D. In the present studies, we have further dissected the epitopes recognized by four monoclonal anti-CD22 antibodies using immunoprecipitation and cross-blocking techniques, immunofluorescence analyses with a variety of cell lines, and immunoperoxidase analyses of 36 normal human tissues. **Two** of the **antibodies**, HD6 and RFB4, have been described previously, and **two**, UV22-1 and UV22-2, are described in this report. Our studies indicate that the four monoclonal **antibodies** show unexpected complexities in their reactivity with **CD22**+ and **CD22**- cells and their reactivity with solubilized **CD22** molecules. The four **antibodies**, which recognize epitopes defined previously as **CD22**-A and **CD22**-B, further subdivide these epitope clusters into four determinants, A1, A2, **B1**, and B2. Furthermore, only **two** of the **antibodies**, RFB4 and UV22-2, are B cell-specific. In summary, our data indicate that RFB4 and UV22-2 would be the **antibodies** of choice for constructing immunotoxins to treat B cell tumors.

9/7/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

07273021 BIOSIS NO.: 000090052902
ANTIBODY L26 RECOGNIZES AN INTRACELLULAR EPITOPE ON THE B-CELL-ASSOCIATED CD20 ANTIGEN
AUTHOR: MASON D Y; COMANS-BITTER W M; CORDELL J L; VERHOEVEN M-A J; VAN DONGEN J J M
AUTHOR ADDRESS: DEP. HAEMATOL., JOHN RADCLIFFE HOSP., OXDORD OX3 9DU, ENGL.
JOURNAL: AM J PATHOL 136 (6). 1990. 1215-1222. 1990.
FULL JOURNAL NAME: American Journal of Pathology
CODEN: AJPAA
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: Monoclonal antibody L26 is a highly selective marker of B cells and B-cell neoplasms in paraffin-embedded tissues, but it suffers from the drawback that the target molecule has not been identified. In this paper we provide evidence by **two** independent techniques that **antibody** L26 recognizes an intracellular epitope on the CD20 antigen (a pan B-cell marker). When this antigen was redistributed on the surface of unfixed viable B cells by incubation with monoclonal anti-CD20 followed by antimouse Ig, the diffuse cytoplasmic staining of L26 was abolished and replaced by coincident dotlike labeling for **antibody** L26 and the CD20 antigen. None of the other **antibodies** tested (covering 10 different B-cell-associated antigens) had this effect on the L26 staining pattern. Furthermore, COS-1 cells transfected with cDNA encoding the **CD20** molecule gave positive staining with **antibody** L26 and with **two** other **CD20** reagents, but not with **antibodies** to other pan B-cell markers (eg, CD19 and **CD22**).

11953443 BIOSIS NO.: 199900199552
Flow cytometric analysis of normal B cell differentiation: A frame of reference for the detection of minimal residual disease in precursor-B-ALL.
AUTHOR: Lucio P; Parreira A; van den Beemd M W M; van Lochem E G; van Wering E R; Baars E; Porwit-MacDonald A; Bjorklund E; Gaipa G; Biondi A; Orfao A(a); Janossy G; van Dongen J J M; San Miguel J F
AUTHOR ADDRESS: (a)Servicio General de Citometria, Laboratorio de Hematologia, Hospital Clinico Universitario, Pase**Spain
JOURNAL: Leukemia (Basingstoke) 13 (3):p419-427 March, 1999
ISSN: 0887-6924
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: During the last two decades, major progress has been made in the technology of flow cytometry and in the availability of a large series of monoclonal antibodies against surface membrane and intracellular antigens. Flow cytometric immunophenotyping has become a diagnostic tool for the analysis of normal and malignant leukocytes and it has proven to be a reliable approach for the investigation of minimal residual disease (MRD) in leukemia patients during and after treatment. In order to standardize the flow cytometric detection of MRD in acute leukemia, a BIOMED-1 Concerted Action was initiated with the participation of six laboratories in five different European countries. This European co-operative study included the immunophenotypic characterization and enumeration of different precursor and mature B cell subpopulations in normal bone marrow (BM). The phenotypic profiles in normal B cell differentiation may form a frame of reference for the identification of aberrant phenotypes of precursor-B cell acute lymphoblastic leukemias (precursor-B-ALL) and may therefore be helpful in MRD detection. Thirty-eight normal BM samples were analyzed with five different pre-selected monoclonal **antibody combinations**: CD10/**CD20**/CD19, CD34/CD38/CD19, CD34/**CD22**/CD19, CD19/CD34/CD45 and TdT/CD10/CD19. **Two** CD19-immature subpopulations which coexpressed B cell-associated antigens were identified: CD34+/**CD22**+/CD19- and TdT+/CD10+/CD19-, which represented 0.11 +- 0.09% and 0.04 +- 0.05% of the total BM nucleated cells, respectively. These immunophenotypes may correspond to the earliest stages of B cell differentiation. In addition to these minor subpopulations, three major CD19+ B cell subpopulations were identified, representing three consecutive maturation stages; CD19dim/CD34+/TdT+/CD10bright/CD22dim/ CD45dim/CD38bright/CD20- (subpopulation 1), CD19+/CD34-/TdT-/CD10+/CD22dim/CD45+/CD38bright/CD20dim (subpopulation 2) and CD19+/CD34-/TdT-/CD10-/CD22bright/CD45bright/CD38dim/ CD20bright (subpopulation 3). The relative sizes of subpopulations 1 and 2 were found to be age related: at the age of 15 years, the phenotypic precursor-B cell profile in BM changed from the childhood 'immature' profile (large subpopulations 1 and 2/small subpopulation 3) to the adult 'mature' profile (small subpopulation 1 and 2/large subpopulation 3). When the immunophenotypically defined precursor-B cell subpopulations from normal BM samples are projected in fluorescence dot-plots, templates for the normal B cell differentiation pathways can be defined and so-called 'empty spaces' where no cell populations are located become evident. This allows discrimination between normal and malignant precursor-B cells and can therefore be used for MRD detection.

13214042 BIOSIS NO.: 200100421191
Effects of specific anti-B and/or anti-plasma cell immunotherapy on **antibody** production in baboons: Depletion of **CD20**- and **CD22**-positive B cells does not result in significantly decreased production of anti-alphaGal **antibody**.
AUTHOR: Alwayn Ian P J; Xu Yuanxin; Basker Murali; Wu Cecelia; Buhler Leo; Lambrigts Denis; Treter Sarah; Harper David; Kitamura Hiroshi; Vitetta Ellen S; Abraham Sonny; Awwad Michel; White-Scharf Mary E; Sachs David H; Thall Aron; Cooper David K C(a)
AUTHOR ADDRESS: (a)Transplantation Biology Research Center, Massachusetts General Hospital, 13th Street, MGH-East, Building 149-9019, Boston, MA, 02129: David.Cooper@tbrc.mgh.harvard.edu**USA
JOURNAL: Xenotransplantation 8 (3):p157-171 August, 2001
MEDIUM: print
ISSN: 0908-665X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Anti-Galalpha1-3Gal **antibodies** (antialphaGal Ab) are a major barrier to clinical xenotransplantation as they are believed to initiate **both** hyperacute and acute humoral rejection. Extracorporeal immunoadsorption (EIA) with alphaGal oligosaccharide columns temporarily depletes antialphaGal Ab, but their return is ultimately associated with graft destruction. We therefore assessed the ability of **two** immunotoxins (IT) and **two** monoclonal **antibodies** (mAb) to deplete B and/or plasma cells **both** in vitro and in vivo in baboons, and to observe the rate of return of antialphaGal Ab following EIA. The effects of the mouse anti-human IT anti-CD22-ricin A (variesCD22-IT, directed against a B cell determinant) and anti-CD38-ricin A (variesCD38-IT, B and plasma cell determinant) and the mouse anti-human anti-CD38 mAb (variesCD38 mAb) and mouse/human chimeric anti-human anti-CD20 mAb (variesCD20 mAb, Rituximab, B cell determinant) on B and plasma cell depletion and antialphaGal Ab production were assessed both in vitro and in vivo in baboons (n=9) that had previously undergone splenectomy. For comparison, two baboons received nonmyeloablative whole body irradiation (WBI) (300 cGy), and one received myeloablative WBI (900 cGy). Depletion of B cells was monitored by flow cytometry of blood, bone marrow (BM) and lymph nodes (LN), staining with anti-CD20 and/or anti-CD22 mAbs, and by histology of LN. EIA was carried out after the therapy and antialphaGal Ab levels were measured daily. In vitro variesCD22-IT inhibited protein synthesis in the human Daudi B cell line more effectively than variesCD38-IT. Upon differentiation of B cells into plasma cells, however, less inhibition of protein synthesis after variesCD22-IT treatment was observed. Depleting CD20-positive cells in vitro from a baboon spleen cell population already depleted of granulocytes, monocytes, and T cells led to a relative enrichment of CD20-negative cells, that is plasma cells, and consequently resulted in a significant increase in antialphaGal Ab production by the remaining cells, whereas depleting CD38-positive cells resulted in a significant decrease in antialphaGal Ab production. In vivo, WBI (300 or 900 cGy) resulted in 100% B cell depletion in blood and BM, >80% depletion in LN, with substantial recovery of B cells after 21 days and only transient reduction in antialphaGal Ab after EIA. variesCD22-IT depleted B cells by >97% in blood and BM, and by 60% in LN, but a rebound of B cells was observed after 14 and 62 days in LN and blood, respectively. At 7 days, serum antialphaGal IgG and IgM Ab levels were reduced by a maximum of 40-45% followed by a rebound to levels up to 12-fold that of baseline antialphaGal Ab by day 83 in one baboon. The results obtained with variesCD38-IT were inconclusive. This may have been, in part, due to inadequate conjugation of the toxin. Cell coating was 100% with variesCD38 mAb, but no changes in antialphaGal Ab production were

observed. variesCD20 mAb resulted in 100% depletion of B cells in blood and BM, and 80% in LN, with recovery of B cells starting at day 42. Adding 150cGy WBI at this time led to 100% depletion of B cells in the BM and LN. Although B cell depletion in blood and BM persisted for>3 months, the reduction of serum antialphaGal IgG or IgM Ab levels was not sustained beyond 2 days. variesCD20 mAb+WBI totally and efficiently depleted **CD20**- and **CD22**-positive B cells in blood, BM, and LN for>3 months in vivo, but there was no sustained clinically significant reduction in serum antialphaGal Ab. The majority of **antibody** secretors are CD38-positive cells, but targeting these cells in vitro or in vivo with variesCD38-IT was not very effective. These observations suggest that **CD20**-and **CD22**-positive B cells are not the major source of antialphaGal Ab production. Future efforts will be directed towards suppression of plasma cell function.

9/7/5 (Item 5 from fil

Immunological detection of minimal residual disease (MRD) in acute lymphoblastic leukemia (ALL): A prospective study in 106 patients.
AUTHOR: Lucio Paulo(a); Silva Maria(a); Faria Teresa(a); Dias Ilidia(a); Santos Ana(a); Santos Isabel(a); Parreira Antonio(a)
AUTHOR ADDRESS: (a)Hematology, Instituto Portugues de Oncologia, Lisbon** Portugal
JOURNAL: Blood 98 (11 Part 1):p459a-460a November 16, 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001
ISSN: 0006-4971
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The detection and quantification of MRD is increasingly important for the clinical care of ALL patients. Flow cytometric detection of MRD was recently standardized by an European BIOMED-1 Concerted Action (Investigation of Minimal Residual Disease in Acute Leukemia: International Standardization and Clinical Evaluation) including laboratories from five different countries. The approach proposed by this cooperative group has not yet been tested in a prospective way. The aim of this study was to evaluate the clinical significance of MRD, as detected by immunophenotypic methods in a population of 106 ALL patients diagnosed at the Instituto Portugues de Oncologia in Lisbon, from January 1994 to December 2000. Sample processing techniques, selection of monoclonal **antibodies**, calibration of flow cytometers, immunophenotypic analysis and criteria for identification of MRD were based on methodologies standardized by the above mentioned cooperative group. Briefly, a direct triple staining technique was used, with the following monoclonal **antibodies** triple **combinations**: CD10/**CD20**/CD19, CD34/CD38/CD19, CD34/**CD22**/CD19, CD19/CD34/CD45 and TdT/CD10/CD19 for B-ALL and CD7/CD5/CD3, CD7/CD4/CD8, CD7/CD2/CD3, CD7/CD38/CD34 and TdT/CD7/cyCD3 for T-ALL. One hundred and six patients with either B-ALL (n=83) or T-ALL (n=23) were included. The median age was 5 years for B-ALL patients (ranging from 1.5 months to 50 y) and 11 years for T-ALL patients (ranging from 18 months to 34 y). The median follow-up was 821 days, ranging from 71 to 2,494. MRD was mesured at the end of induction treatment, and patients were stratified in three groups, according to the number of blast cells identified in the post-induction bone marrow samples: MRD- (less then 0.01% blasts); MRD+ (between 0.01% and 1% blasts) and persistent disease (PD) (blasts over 1%). At the end of the study, 70 out of 106 patients (66%) were in continuous complete remission, while 36 (34%) relapsed. In order to identify the patients's clinical and laboratorial characteristics that could be related with relapse, we evaluated the impact of age, sex, race, WBC counts at diagnosis, cell lineage of leukemic cells, ploidy index, cytogenetics and MRD found in bone marrow after induction therapy. Multivariate Cox Regression analysis showed no association between relapse and race, sex, WBC counts at diagnosis, cell lineage and ploidy index (pgtoreq0.05). In contrast, a statistically significant association was found between relapse and the presence of chromosome aberrations such as t(9;21) and t(4;11) (p=0.048), age (pltoreq0.001) and MRD+ or PD after induction therapy (pltoreq0.001). The correlation of MRD+ or PD with the risk of relapse was observed both in T (p=0.03) and B-ALL (pltoreq0.001) as well as in adults aged over 16 y (pltoreq0.001) and in the pediatric population (p=0.003). In this study, we were able to demonstrate that using a simple, fast and standardized method for MRD detection, the size of the leukemic residual population after induction therapy is evaluable in all ALL patients and has important prognostic implications.

13501055 BIOSIS NO.: 200200129876
Antitumour activity of Calicheamicin theta, Doxorubicin and anti-CD19 immunoconjugates in a human pre-B ALL cell line.
AUTHOR: Jendreyko Nina(a); Bernt Kathrin(a); Gaedicke Gerhard(a); Wrasidlo Wolfgang(a); Beutler Ernest
AUTHOR ADDRESS: (a)Dept. of Pediatrics, Charite, Humboldt University, Berlin**Germany
JOURNAL: Blood 98 (11 Part 1):p105a November 16, 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001
ISSN: 0006-4971
RECORD TYPE: Abstract

(FILE 'HOME' ENTERED AT 17:56:03 ON 12 APR 2002)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 17:56:22 ON 12 APR 2002

```
L1          88834 S (SLE OR LUPUS)
L2           3141 S CD22
L3           2942 S "B CELL DEPLETION" OR "B LYMPHOCYTE DEPLETION" OR (DEPLET?
(4
L4             63 S L1 AND L3
L5             29 DUP REM L4 (34 DUPLICATES REMOVED)
L6              1 S L5 AND L2
L7             46 S L1 AND L2
L8             25 DUP REM L7 (21 DUPLICATES REMOVED)
L9         169547 S AUTOIMMUNE
L10           177 S L9 AND L3
L11            80 DUP REM L10 (97 DUPLICATES REMOVED)
L12             2 S L11 AND L2
L13            13 S L11 AND L1
```

L5 ANSWER 2 OF 29 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002109883 MEDLINE
DOCUMENT NUMBER: 21830488 PubMed ID: 11841453
TITLE: Anti-CD20 monoclonal antibody (Rituximab) for life-threatening autoimmune haemolytic anaemia in a patient with systemic **lupus** erythematosus.
AUTHOR: Perrotta Silverio; Locatelli Franco; La Manna Angela; Cennamo Lucia; De Stefano Piero; Nobili Bruno
CORPORATE SOURCE: Department of Paediatrics, 2nd University of Naples, Naples, Italy.. silverio.perrotta@unina2.it
SOURCE: BRITISH JOURNAL OF HAEMATOLOGY, (2002 Feb) 116 (2) 465-7.
Journal code: 0372544. ISSN: 0007-1048.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020214
Last Updated on STN: 20020326
Entered Medline: 20020325

AB Innovative approaches are needed for patients with systemic **lupus** erythematosus (**SLE**) who develop autoimmune haemolytic anaemia (AIHA) that does not respond to conventional treatment. Rituximab, a chimaeric anti-CD20 monoclonal antibody, has been demonstrated to be highly effective for in vivo **B-cell depletion** . We report an 18-year-old-girl with **SLE** and life-threatening AIHA that did not respond to steroids, intravenous immunoglobulin and cyclosporin A. Rituximab was given weekly at 375 mg/m2 for two doses. The drug was well tolerated and the patient had no adverse effects. Her haemolytic disorder markedly ameliorated, with a progressive increase of haemoglobin levels, starting a few days after therapy. The patient remains disease-free 7 months later.

L8 ANSWER 10 OF 25 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:814347 CAPLUS
DOCUMENT NUMBER: 133:361915
TITLE: Treatment of autoimmune diseases with antagonists which bind to B cell surface markers
INVENTOR(S): Curd, John G.; Kunkel, Lori A.; Grillo-Lopez, Antonio J.
PATENT ASSIGNEE(S): Genentech, Inc., USA; Idec Pharmaceuticals, Inc.
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000067796	A1	20001116	WO 2000-US40018	20000504
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1176981	A1	20020206	EP 2000-928991	20000504
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 2000011197	A	20020219	BR 2000-11197	20000504
NO 2001005417	A	20020107	NO 2001-5417	20011106

PRIORITY APPLN. INFO.:

US 1999-133018P	P	19990507
US 1999-139621P	P	19990617
WO 2000-US40018	W	20000504

RE
(1) Johnston, P; BLOOD, PART 2 1999, V94(10, SUPP 1), P4386
(2) Lee, E; BLOOD 1998, V92(9), P3490 CAPLUS
(3) Mow, B; BLOOD, PART 2 1999, V94(10, SUPP 1), P3526
(4) Scheuermann, R; US 5686072 A 1997 CAPLUS
(5) Univ Leland Stanford Junior; WO 9503770 A 1995 CAPLUS

L8 ANSWER 20 OF 25 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:40319 CAPLUS
DOCUMENT NUMBER: 126:73466
TITLE: Tumor necrosis factor-.alpha. (TNF-.alpha.) and interleukin-6 (IL-6) in B-lymphocyte function
AUTHOR(S): Rieckmann, P.; Tuscano, J. M.; Kehrl, J. H.
CORPORATE SOURCE: Dep. Neurology, Julius-Maximilians Univ., Wuerzburg, D97080, Germany
SOURCE: Methods (San Diego) (1997), 11(1), 128-132
CODEN: MTHDE9; ISSN: 1046-2023
PUBLISHER: Academic
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 65 refs. Two cytokines important in the regulation of B-cell function are tumor necrosis factor-.alpha. (TNF-.alpha.) and interleukin-6 (IL-6). They act at different steps in B-cell differentiation and can be produced by the B cells themselves upon appropriate stimulation. Crosslinking of surface Ig and signaling through **CD22** or CD40 lead to increased of both cytokines. Neutralization of TNF-.alpha. or IL-6 biol. activity in B-cell cultures results in a significant redn. in B-cell proliferation and Ig secretion. Increased prodn. of these cytokines is found in several diseases assocd. with aberrant B-cell function. This review will focus on the role of TNF-.alpha. and IL-6 in normal and pathophysiol. conditions of B-cell function.

L12 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:935450 CAPLUS
DOCUMENT NUMBER: 136:68707
TITLE: Treatment of B-cell associated diseases such as malignancies and **autoimmune** diseases using a cold anti-CD20 antibody/radiolabeled anti-**CD22** antibody combination
INVENTOR(S): White, Christine
PATENT ASSIGNEE(S): Idec Pharmaceuticals Corporation, USA
SOURCE: PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001097858	A2	20011227	WO 2001-US18939	20010614
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002039557	A1	20020404	US 2001-883962	20010620

PRIORITY APPLN. INFO.: US 2000-212668P P 20000620

AB Treatment of B-cell assocd. diseases including **autoimmune** and B-cell malignancies such as leukemias, lymphomas, using the combination of an anti-CD20 antibody, preferably RITUXAN.RTM. and a radiolabeled anti-**CD22** antibody, preferably an 90Y labeled humanized anti-**CD22** antibody, is described. These therapeutic regimens provide for enhanced **depletion** of **B cells**, and therefore reduce the risk in B cell malignancy treatment of relapse assocd. with RITUXAN.RTM. and, moreover, provide for prolonged immunosuppression of B-cell immune responses, esp. in the context of **autoimmune** diseases and transplant.